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MODULE 7
PRINCIPLES OF OCCUPATIONAL MEDICINE

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Disclosure: Capt Fajardo does not have any financial arrangements or affiliations with any corporate organizations that might constitute a conflict of interest with regard to this continuing education activity.

Goals:

1. Understand the principles of Occupational Medicine
2. Learn the importance of taking an occupational medical history.
3. Learn the health effects of chemical exposures.
4. Understand the historical perspective of Occupational Medicine.

As part of this lecture the reader should become familiar with the following reading materials, included as part of this presentation:
"Taking an Exposure History" from the Agency for Toxic Substances and Disease Registry (ATSDR).
"Medical Management Guidelines for Acute Chemical Exposures" from the Agency for Toxic Substances and Disease Registry (ATSDR). Section 1- General Information and section 2- Health Effects.

Note: The Agency for Toxic Substances and Disease Registry (ATSDR) is an agency of the Public Health Service in the U.S. Department of Health and Human Services. The mission of the ATSDR is to prevent or mitigate adverse human health effects and diminished quality of life resulting from exposure to hazardous substances in the environment. In pursuit of this mission, ATSDR provides educational and referral resources to health care providers who are responsible for chemically exposed patients.

Excerpts from ATSDR's educational series are presented here as part of this educational training module. The aforementioned sections describe the differences between primary and secondary contamination. They also describe the physical characteristics of chemical agents, describe the most common routes of exposure and detail the health effects of chemical agents on the various organ systems.
The monograph on "Taking an Exposure History" presents case studies as it illustrates the importance of the occupational and environmental health history. It also provides important information on the most common toxicants found in the home/environment.

The knowledge gained from this training module and accompanying reading materials will serve to enhance the abilities of primary health care providers to adequately diagnose

occupational health diseases by adequately detecting the effects of chemical exposures on the various organ systems.

PRINCIPLES OF OCCUPATIONAL MEDICINE

The history of Occupational Medicine dates back to ancient times but it was not until Bernadina Ramazzini (1633-1714), recognized by many as the father of Occupational Medicine, that attention was given to diseases caused by the workplace environment. This Italian epidemiologist performed studies on causality by following workers into the mines and factories to experience the conditions under which they worked. Ramazzini would advise health providers on the importance of inquiring about the occupation of their workers in addition to usual health questions. Worksite visits and a general understanding of the working conditions are an essential part of Occupational Medicine. Knowledge of occupational exposures can also be traced into antiquity and industrial hazards have been known for centuries. The fact is chemicals are a part of our every day lives and thousands of these products, in their natural form, can be found in our environment.

A brief historical perspective on the actions of chemicals follows. Symptoms of lead intoxication can be traced to the first century A.D., a condition that befell many of the Roman emperors and higher aristocracy. Roman emperors such as Nero and Claudius were among those reported to have demonstrated signs of neurotoxicity. Claudius reportedly developed slurred speech, slobbered and had a staggering gait; Nero played the fiddle while Rome burned and eventually became insane. These effects were presumed to be secondary to the neurotoxic effects of metals ingested with their lavished meals, served in bronze and copper pots, and their lead-treated wines. Roman emperors reportedly may have consumed up to a gram of lead per day. Mercury intoxication was reported in France in the seventh century. Mercury was commonly used to stiffen the brims of hats causing intoxication on workers within the hat industry-making the expression "mad as a hatter" a colloquial term. This was also depicted in the story of "Alice in Wonderland". In the late 1700's Sir Percival Pott discovered the association between coal tar exposure and the development of cancer in chimney sweeps. The participant is encouraged to seek further reading on these historical events by reading any one of the following:

"Lead Poisoning and the Fall of Rome" by S.C. Gilfillan. Journal of Occ. Med. (1965) 7:53-60.

"Lead Poisoning in the Ancient World" by H.A. Waldron. Medical History. (1973) 17:391-399.

"Lead and Lead Poisoning in Antiquity" by J.O. Nriago. John Wiley and Sons, New York (1983).

Hazardous exposures remain as much a problem today as they did in the 1700's. Due to the severity of the events exposure from industrial agents such as benzene, asbestos, polychlorinated biphenyls, and vinyl chloride are commonly reported. Yet, in our everyday activities, we come in contact with toxic substances such as gasoline, pesticides and a number of household products such as chlorine, ammonia and disinfectants, with the potential for disease or injury. The commonly brief exposure to these agents

ordinarily does not result in incapacitation or disease. As a result, we often disregard the precautions that accompany the use of these products.
Hazardous substances are defined in the Comprehensive Environmental Response Compensation and Liability Act (CERCLA) as any chemical regulated by the Clean Air and Water Act. A hazard, on the other hand, requires knowledge of the use of the substance. One must understand the inherent ability of the substance to do harm and the ease by which the substance can affect a normally safe working environment.
CERCLA requires an annual evaluation of substances from a priority list of 275 agents. From this evaluation ATSDR publishes a list called the "Top 20 Hazardous Substances". This list includes the following products:

1. Arsenic
2. Lead
3. Mercury
4. Vinyl chloride
5. Benzene
6. Polychlorinated Biphenyls
7. Cadmium
8. Benzo(a)pyrene
9. Benzo(b)fluoranthene
10. Polycyclic aromatic hydrocarbons
11. Cloroform
12. Aroclor 1254
13. DDT
14. Aroclor 1260
15. Tricloroethylene
16. Chromium (+6)
17. Dibenz(a,h)anthracene
18. Dieldrin
19. Hexachlorobutadiene
20. Chlordane

In the military environment exposure to hazardous substances may also occur as a result of combat. Weaponized hazardous substances are expected to play a major role in future military conflicts with the intent of degrading unit effectiveness. Most military medical providers are not familiar with the effects and/or management of these substances, a fact that was apparent during the initial stages of Operation Desert Storm/Desert Shield. As with all other previous national conflicts, the U.S. Coast Guard and the Public Health Service had an important though limited presence in that conflict. In the future Coast Guard medical officers may be required to provide care to personnel involved in such missions or find themselves as active participants in similar situations or as part of humanitarian efforts. One must not forget the ever-present threat from bioterrorism. A terrorist act can occur in any city or military installation. Awareness of the effects of toxic substances and learned medical defenses can go a long way to minimize mortality or morbidity. Chemical warfare agents will not be discussed in any greater detail. Medical providers, however, are urged to visit the Virtual Naval Hospital web site at: www.vnh.org and seek further reading on "First Aid in Toxic Environments" and "Medical Management of Chemical Casualties Handbook".

Occupational Medicine encompasses a number of health services. It includes baseline examinations, periodic evaluations, fitness for duty determinations and disability assessments. It also includes knowledge of the occupational and nonoccupational environment in order to provide a safer workplace, health promotion and assuring adequate medical care and rehabilitation. The goal should always be on promoting the worker's health.

There is a long held belief that occupational diseases are somehow different than those commonly encountered in general practice. However, occupational cancers are indistinguishable from cancers of nonoccupational origin except by a detailed history of exposure to a carcinogenic agent confirming the association. Only a minority of occupational diseases, such as those caused by heavy metal affliction, present with sufficient evidence that they can be identified through routine testing procedures.

Medical practitioners should always consider the possibility that nonoccupational conditions might have a synergistic and possibly enhancing effect on an occupational disease process. Disease causing agents have varied mechanisms of action and as a result different intervals for onset of symptoms. Some agents produce their effects soon after exposure and thus their causal relation to the disease process can be identified. Other agents trigger a delayed immune reaction and their effects are not realized for months or years after the exposure. Yet, others have a long latency for disease development and clinical symptoms may not become apparent for many years. The latter effect is of great concern within our organization, as Marine Safety personnel are occasionally exposed to agents such as Asbestos and Benzene that exhibit this characteristic.
It is important to also note the relationship that exists between dose exposure and subsequent effect. Generally speaking, the higher the exposure the greater the likelihood of being affected and subsequently the more serious the effects. Individuals however, differ in their response to environmental exposures. This may be due to genetic factors, differences in age, gender or even underlying medical conditions. This variability may obscure the relationship between the health effects and the environmental exposures. Medical providers should learn to recognize the varying patterns of disease development and the difficulties this may present in the timely identification of a hazardous substance and its effects on an at-risk population.

THE OCCUPATIONAL HEALTH HISTORY

The occupational health history is an integral part of the assessment of work related problems. The evaluation of a worker, potentially exposed to industrial hazards, can be obtained through various mechanisms. The approach chosen for the Occupational Medical Surveillance and Evaluation Program is to perform a thorough initial evaluation that is repeated every five years of employment followed by brief periodic examinations. The information sought during the occupational history differs from the one obtained during a general health examination. For this reason, the INITIAL OMSEP examination asks similar questions in both the CG 5447 and the SF 93. The medical practitioner should be aware of the differences between the information obtained as part of the environmental health history from that obtained in a general clinical setting. For example, a worker responding to questions about respiratory problems may think about head colds, sinus conditions and ear infections. Similar questions in the setting of the occupational history may elicit responses about respiratory wear devices or sensitizing agents. Additionally, the occupational history has several purposes:

1. Screening. By screening for previously known exposures, such as Asbestos, a counseling program on tobacco cessation can be provided that would illustrate the carcinogenic potential of these products. It also prevents the aggravation of

underlying medical conditions by restricting exposure to substances that could aggravate existing symptomatology.

2. Awareness. Through accurate questioning about occupational factors hazardous situations can be identified and steps taken to mitigate further risks. Alluding to the risks of exposures during the questioning process can enhance utilization of safety equipment.
3. Diagnosis. Understanding a worker's past medical history, may serve to provide a more accurate diagnosis. Knowledge of the worker's drinking (ETOH) habits, use of medications and surgical history may explain laboratory abnormalities, radiological findings and clinical manifestations.
4. Disability. Adequate recording and reporting of workplace exposures, work limitations and other occupational related factors are essential in the assessment of future work limitations and potential compensation. Failure to document suspected acute exposures, especially on HAZMAT (Strike Team) members and firefighters, create difficulties in corroboration of suspected occupationally related events later in life.
5. Communication. The history taking process is the best mechanism to learn about the worker's non-occupational and associated environmental history. It is the mechanism to establish a rapport with the individual that could lead to a more relaxed and comprehensive discussion and understanding of work, social, emotional and psychological issues affecting the health of the individual.

The worker has the best knowledge of past and present exposures. Many workers are well informed about exposure agents and can often relate symptoms or signs that shed light on undiagnosed or missed conditions. The main goal of an occupational health examination is to gain a thorough and comprehensive history of occupational and environmental factors that could be playing a role in the development of a related disease process. Questions about the health of other workers, similarity of complaints or symptoms, can lead to the identification of an occupational disease process. Identifying other workers with similar complaints maybe the key to a lifesaving diagnosis. Care most be provided, however, not to violate the privacy of the other individuals.

A general workplace assessment, via the use of questionnaires, can provide access to a group of workers without singling out any one particular individual. Inquiring about conditions as they relate to a workday verses a non-work day; a weekend or vacation day as well as particular days of the week may shed some light as to the presence of an underlying problem. In the Coast Guard setting a medical officer can rely on the Safety and Environmental Health Officer to provide this information. The SEHO is responsible for worksite monitoring and can perform qualitative or quantitative studies that might help identify the presence of a hazardous substance.

The attached required reading, compliments the occupational history guidelines and required forms found in Chapter 12 of the Coast Guard's Medical Manual, COMNDTINST M6000 series. After completing all required reading material the participant should complete the accompanying post-test in order to obtain continuing medical education credits.

General Information Section

Chemical Name (Chemical Formula)

CAS Number; UN Number

Common synonyms

- **Potential for secondary contamination**
- **Common form and important characteristics such as odor and flammability**
- **Routes of exposure and potential for absorption**

Secondary Contamination

Primary contamination refers to direct contact of the victim with the hazardous material. Secondary contamination refers to the transfer of material from the victim to personnel or equipment. The potential for secondary contamination has implications for decontamination and triage of victims and for the protection of rescue and health care personnel. Immediate victim decontamination is recommended for materials that pose risks of secondary contamination; this eliminates both the potential for rescuer contamination and further exposure to the victim.

A substance poses a risk of secondary contamination if it is both toxic *and* likely to be carried on the clothing, skin, or hair of victims in sufficient quantities to threaten other personnel. Substances that present the most serious risks of secondary contamination include the following:

- highly toxic liquids and solids or finely divided solids (e.g., organophosphate pesticides)
- radioactive liquids and dusts
- certain biologic agents (e.g., harmful viruses or bacteria)

Every effort must be made to decontaminate contaminated victims before they are transported to a medical care facility.

Examples of substances with little or no risk of secondary contamination include the following:

- gases (e.g., carbon monoxide, arsine)
- vapors (unless they condense to a liquid state on clothing or skin)
- substances with no serious toxicity or skin absorption (e.g., propylene glycol, motor oil)

Note that although several of the substances listed above are highly toxic (e.g., arsine, carbon monoxide), they do not pose risks of secondary contamination because these chemicals will not contaminate the victim; therefore, they cannot secondarily contaminate rescuers.

Secondary contamination also may be a risk in cases of ingestion. Ingested materials may react with stomach acid to produce noxious gases, which can pose risks to both the victim and rescuers. Vomitus may off-gas the hazardous material or a reaction product. Toxic vomitus should be quickly isolated in closed containers.

Previously published documents on hazardous materials have recommended zipping patients into body bags to minimize the transfer of chemical from patient to rescuer. This technique is not completely effective for preventing rescuer exposure, and it may pose a significant risk of increased dermal absorption to victims. Body bags are not recommended as an alternative to thorough decontamination.

Description

This section summarizes the color, odor, and physical state (solid, liquid, or gas) of the chemical at room temperature. Methods of shipment or storage and the physical hazards associated with the chemical are also described.

Routes of Exposure

The most likely routes of exposure—inhalation, direct contact with the skin or eyes, and ingestion—are described. For each route of exposure, the risk of injury depends on the toxicity of the chemical involved, the concentration of the material, and the duration of contact.

Inhalation

Inhalation is the most common route of exposure to gases and vapors. Liquids and solids may also be inhaled when they are finely divided mists, aerosols, fumes, or dusts. Highly water-soluble gases and vapors and larger mist or dust particles (greater than 10 microns in diameter) generally are deposited in the upper airways. Less soluble gases and vapors and smaller particles can be inhaled more deeply into the respiratory tract. Usually, highly water-soluble materials rapidly produce symptoms of upper-airway irritation, whereas less soluble materials may produce delayed symptoms in the lower airways. Inhaled substances may be absorbed into systemic circulation, causing toxicity to various organ systems. When available, information is provided on odor threshold, warning properties, and symptoms to be expected at specific exposure levels.

Skin/Eye Contact

Skin and eye contact can occur by exposure to solids, liquids, or gases. Corrosive agents cause direct damage to tissues by various mechanisms including low or high pH, chemical reaction with surface tissue, removal of normal skin fats (defatting), or removal of moisture (desiccant effect). Some chemicals absorbed through the skin and eyes can produce systemic toxicity. Absorption, and therefore toxicity, is more likely to occur when the normal skin barrier is disrupted (e.g., chemical burn, cut, or abrasion) or when the chemical is highly fat-soluble (e.g., organophosphate and organochlorine pesticides).

Ingestion

Ingestion is not a common route of exposure in most hazardous materials (HAZMAT) incidents, although it is common in suicide attempts. Ingestion of corrosive agents can cause severe burns to the mouth, throat, esophagus, and stomach. Ingested chemicals may also be aspirated into the lungs, especially after vomiting, causing chemical pneumonitis. Ingested chemicals may react with stomach acid, creating products that are toxic to the patient, and potentially, the health care provider (e.g., hydrogen cyanide from ingested cyanide salts).

Sources/Uses

This section describes the chemical's most common uses and the methods of production.

Standards and Guidelines

Government agencies and professional organizations have established standards and guidelines for hazardous chemical exposures. The standards and guidelines address both acute and chronic exposures.

The Occupational Safety and Health Administration (OSHA) **permissible exposure limit** (PEL) is a regulatory limit established to avoid adverse health effects from exposure. PELs are time-weighted-average (TWA) air concentrations. In most cases, a healthy, working adult can be exposed to a chemical at the PEL for an 8-hour workday and a 40-hour workweek and suffer no adverse health effects. If the measured air concentration at a HAZMAT incident is less than the PEL and the exposure is short-term, persons at the scene are probably not at serious risk. The OSHA "skin" designation indicates the likelihood of dermal absorption.

The **OSHA ceiling** is an instantaneous concentration that must not be exceeded any time. If instantaneous monitoring is not feasible, the ceiling is normally assessed as a 15-minute TWA concentration.

The **OSHA short-term exposure limit (STEL)** is a 15-minute (unless otherwise noted) TWA concentration that should not be exceeded at any time, even if the 8-hour TWA concentration is below the PEL.

The National Institute for Occupational Safety and Health (NIOSH) recommends workplace exposure guidelines. The **NIOSH immediately dangerous to life or health (IDLH)** level represents the maximum chemical concentration from which one could escape within 30 minutes without a respirator and without experiencing any escape-impairing (e.g., severe eye irritation) or irreversible health effects.

The American Industrial Hygiene Association (AIHA) **Emergency Response Planning Guidelines (ERPG)** state concentrations at which one might reasonably anticipate observing adverse effects from exposure to specific substances. Unlike occupational exposure standards, ERPG levels are applicable to the general public, including children and the elderly. The three ERPG levels vary with the health effects expected with exposure (transient symptoms, ability impairment, and life-threatening) and apply to practically all persons. The table in the *Properties* section includes only ERPG-2–the exposure level that could impair a person's ability to take protective action.

ERPG levels are defined as follows:

ERPG-1 is the maximum airborne concentration below which it is believed that nearly all persons could be exposed for up to 1 hour without experiencing symptoms other than mild transient adverse health effects or perceiving a clearly defined objectionable odor.

ERPG-2 is the maximum airborne concentration below which it is believed that nearly all persons could be exposed for up to 1 hour without experiencing or developing irreversible or other serious health effects or symptoms that could impair their abilities to take protective action.

ERPG-3 is the maximum airborne concentration below which it is believed that nearly all individuals could be exposed for up to 1 hour without experiencing or developing life-threatening health effects.

Physical Properties

Description: Physical state and useful characteristics of the chemical at room temperature are presented.

Warning properties: Odor and irritation are the primary determinants of exposure awareness. When available, an objective description of odor (e.g., garlic-like) and the lowest air concentration that can be detected (i.e., odor threshold) is provided. For chemicals with an odor threshold below the toxic air concentration, odor may provide an adequate warning of dangerous exposure conditions. However, a chemical is considered to have inadequate warning properties if it has no detectable odor at toxic air concentrations, has an odor that is not reliably detected because of olfactory fatigue, or does not cause irritation.

Molecular weight (MW) is the sum of the weights of the atoms in a molecule. Molecular weight is provided in daltons, a unit that is based on the mass of oxygen-16. Molecular weight can be used to convert measurements of air concentrations of chemicals from parts per million (ppm) to milligrams per cubic meter (mg/m^3) using the following equation:

$$mg/m^3 = (ppm \times MW) / 22.4\ L/mole$$

(1 mole of gas occupies 22.4 L at standard temperature and pressure).

Boiling point of a liquid is the temperature at which its vapor pressure is equal to the atmospheric pressure. A boiling point at or below room temperature means that the chemical is in the gaseous state at room temperature.

Freezing point is the temperature at which a chemical's solid phase is in equilibrium with the liquid phase. Freezing point and melting point are equal in numeric value. The term "freezing point" refers to the temperature at which a liquid forms a solid; "melting point" refers to the temperature at which a solid forms a liquid.

Specific gravity is the ratio of the density of a liquid to the density of a reference material (usually water). A specific gravity less than 1 indicates that the substance will float on water; a specific gravity greater than 1 indicates that the substance will sink in water.

Vapor pressure is the pressure (expressed in millimeters of mercury [mm Hg]) of a vapor in equilibrium with its liquid or solid form at a given temperature. The higher the vapor pressure, the greater the amount of chemical existing in the vapor phase. A vapor pressure greater than 760 mm Hg at room temperature indicates that the chemical exists as a gas.

Gas density is the ratio of the density (weight per volume) of a substance (at a given temperature) to the density of air (at that temperature). A gas density greater than 1 indicates that the vapor or gas is heavier than air. A gas heavier than air may collect in low-lying areas where it can displace air, creating an oxygen-deficient atmosphere.

Water solubility indicates the degree to which a substance dissolves in water at a specific temperature. Water solubility is measured in weight of substance per volume of water (e.g., g/100 ml or %). Water solubility may indicate the effectiveness of water in decontamination. A substance that is water soluble is likely to be removed from the skin and hair with a plain water wash. Substances that are poorly water soluble may require the use of soap.

Flammability is the ease with which a material will ignite. Flammable chemicals have flashpoints below 100°F; combustible chemicals have flashpoints between 100°F and 200°F; and nonflammable chemicals have flashpoints above 200°F.

Flammable range (lower explosive limit to upper explosive limit) is expressed as the percentage of gas or vapor dispersed in air that will burn when an ignition source is present. The temperature, the flammable range, and the potential for a vapor or gas to travel to an ignition source and flash back may affect rescue activities. The flammable range may indicate the need for special protective clothing. Most chemical-resistant protective clothing is neither heat- nor flame-resistant and may melt if a fire occurs.

Incompatibilities

HAZMAT incidents commonly involve more than one chemical. Incompatibility and reactivity information, primarily from the *NIOSH Pocket Guide to Chemical Hazards*, is included in this section.

Health Effects Section

- **Common symptoms**
- **Systemic effects and mechanism of action**

Health risk depends on the intrinsic toxic potential of the chemical, its concentration, and the duration of exposure. Highly toxic chemicals may pose a risk of illness even if the exposure duration is brief or the concentration of the substance is low. Even mildly toxic substances, however, can be hazardous if the exposure is prolonged or the concentration is high.

Acute Exposure

When suspected or known, the mechanism of action is discussed.

Acute exposure is defined as chemical exposure of less than 14 days duration. Most HAZMAT incidents involve acute exposures that last only minutes, but the chemical concentration may be extremely high. Although HAZMAT incident exposures are likely to be short, risks of adverse health effects still exist. The onset of health effects caused by acute exposure can be immediate or delayed.

Organ systems or metabolic processes that are adversely affected by the chemical are discussed in the following sections. The organ system or metabolic process mentioned first in each chemical protocol is the most severely affected; those not affected by the chemical are not addressed.

Cardiovascular

Many chemicals have direct depressant or stimulatory effects on cardiac function. Hypotension and dysrhythmias may be aggravated by hypoxia from respiratory depression or pulmonary aspiration of gastric contents. Hypotension may also occur because of volume depletion from excessive vomiting, diarrhea, or severe chemical burns.

Certain solvents (e.g., chlorinated hydrocarbons, freons, aromatic hydrocarbons) may lower the myocardial threshold to the dysrhythmogenic effects of catecholamines. For several hours after solvent exposure, a victim may be susceptible to ventricular dysrhythmias (e.g., premature ventricular contractions, ventricular tachycardia, or ventricular fibrillation) especially from administered sympathomimetic drugs such as bronchodilators or dopamine or the increased quantity of endogenous epinephrine produced during intense physical activity.

CNS

Central nervous system (CNS) depressants (e.g., hydrocarbon solvents) cause a generalized decrease in brain activity. Headache, dizziness, confusion, lethargy, stupor, or coma may result. Severe depression of the brain stem can cause respiratory arrest and cardiovascular collapse. Some chemical depressants have early stimulatory effects, producing euphoria and giddiness similar to ethanol.

CNS stimulants (e.g., organophosphate insecticides) can cause agitation, anxiety, delirium, and seizures. Excessive muscular activity associated with seizures can cause hyperthermia.

Dermal

Dermal contact with chemicals can produce local injury; if absorbed, chemicals can also produce systemic effects. Local injuries (e.g., burns from mineral acids) usually are immediately obvious. However, a few chemicals (e.g., alkaline corrosives, hydrofluoric acid) cause a progressive penetrating injury that may not be apparent for hours.

The skin generally provides a relatively impermeable protective barrier. Many chemicals disrupt the skin's integrity by removing fats, producing chemical burns, or destroying cells. Physical injury such as thermal burns or traumatic events may also result in loss of the skin's barrier effect. Disruption of the normal protective barrier allows easier entry of chemicals into systemic circulation. Systemic illness can also occur without skin damage because many fat-soluble chemicals (e.g., some organophosphate insecticides) rapidly penetrate intact skin.

Electrolyte

Some chemicals can produce effects on serum electrolytes (e.g., potassium, calcium, sodium) and total anion gap. Electrolyte imbalance can cause muscle weakness and cardiac dysrhythmias.

Gastrointestinal

Nausea, vomiting, abdominal pain, and diarrhea are common symptoms after chemical exposure and may be due to direct gastrointestinal irritation or to systemic effects. Ingestion of some chemicals can also cause severe corrosive injury to the mouth, throat, esophagus, and stomach, with bleeding, perforation, scarring, or stricture formation as potential sequelae.

Hematologic

Components of the blood and blood-forming organs can be damaged by many chemicals (e.g., arsine, benzene). Most hematologic changes (e.g., hemolysis, methemoglobinemia, bone marrow suppression, and anemia) can be detected by blood tests or simply by the color or appearance of the blood.

Hepatic

Some chemical exposures result in acute injury to the liver, which typically does not manifest for 2 to 3 days after exposure. At that time, laboratory tests will show abnormal liver function (e.g., elevated bilirubin or aminotransferase levels or increased prothrombin time). Toxic hepatitis may progress to liver failure and death.

Immunologic

Immunologic effects may include induced sensitivity and allergy.

Metabolic

Metabolic acidosis is the most common adverse metabolic effect that occurs after chemical exposure. Acidosis results from an accumulation of acid anions such as formic, lactic, or oxalic acid.

Musculoskeletal

Musculoskeletal damage due to chemical exposure is unusual. Some effects are arthritis and hardening, destruction, or cancer of the bone.

Ocular

Most serious ocular injuries result from direct eye contact with corrosive liquids or solids. High concentrations of or prolonged exposures to gases or vapors may also injure the eye. Severe eye exposure carries a risk of blindness or other visual impairment and demands immediate evaluation by an ophthalmologist.

Most patients who have eye injuries involving the conjunctival or corneal surfaces experience pain and irritation, excessive lacrimation, and possibly crusting and swelling of the eyelids. Generally, corneal damage causes intense pain and the sensation of a foreign body in the eye.

Peripheral Neurologic

Peripheral nervous system effects can include changes in sensation, reduced reflexes, and impaired motor function. Effects are pronounced in the largest muscle groups such as those in the lower limbs.

Renal

Some chemicals injure the kidneys directly. In addition, any poisoning causing massive muscle destruction can lead to kidney injury from excessive myoglobin in the kidney tubules.

Respiratory

Inhalation of a chemical irritant (e.g., ammonia, chlorine) usually causes rapid onset of burning and irritation of the nose, throat, and upper respiratory tract. Painful coughing, wheezing, and stridor may develop. If the exposure is massive, death may rapidly ensue from upper airway obstruction, massive alveolar destruction, or asphyxiation. Chest radiography may indicate pulmonary edema when damaged lung cells allow fluid to leak into the alveoli (referred to as

noncardiogenic pulmonary edema because the fluid accumulation is not caused by left ventricular failure, which occurs in cardiogenic pulmonary edema).

The onset and location of respiratory symptoms is partially related to the water solubility of the inhaled chemical. Highly water-soluble gases, such as ammonia, cause rapid onset of symptoms (burning nose and throat, painful cough, stridor, wheezing) as the gases dissolve in the mucous membranes of the upper respiratory tract. However, less soluble gases such as phosgene are breathed deeply into the lower airways and typically cause only mild or no early symptoms; noncardiogenic pulmonary edema may develop after 12 to 36 hours.

Injury to the respiratory tract also can occur after ingestion of a chemical substance. The unconscious or convulsing patient may vomit and then, because of depressed airway protective reflexes, may aspirate gastric contents into the lungs. Pulmonary aspiration of an ingested hydrocarbon can cause severe pneumonitis. Hydrocarbons irritate the lung tissue and interfere with surface tension in the alveoli, disrupting gas exchange. Pulmonary aspiration can sometimes be prevented by inserting a cuffed endotracheal tube into the patient's airway or by placing the patient in a head-down, left-side position and using suction immediately if vomiting occurs.

Potential Sequelae

Known or suspected sequelae and the prognosis for recovery after an acute exposure are described in this section. Signs and symptoms expected at various stages of recovery and the potential for permanent deficits are presented.

Chronic Exposure

Repeated, low-level exposures, typically over a long period of time, may produce health effects that differ in type or degree from effects of acute exposure. Most information about chronic toxicity is from epidemiologic studies and case reports of workplace exposures. Because HAZMAT incidents are unlikely to involve repeated or long-term exposures, chronic health effects are outlined only briefly.

Some major concerns of patients who have experienced an acute chemical exposure are the risks of cancer, reproductive effects, or impaired fetal development. No data exist on these outcomes from acute exposure to most chemicals. However, to guide the clinician who must address these patient concerns, we have included carcinogenic, reproductive, and developmental effects

that have resulted from chronic exposure to the chemical. It is not known whether the data from chronic exposures are applicable to victims who are acutely exposed in a HAZMAT incident.

Carcinogenicity

The cancer information included in this section is derived from assessments made by the Department of Health and Human Services (DHHS), the International Association for Research on Cancer (IARC), or the Environmental Protection Agency (EPA). These organizations develop ratings of chemicals that indicate the cancer-producing ability of the chemicals. The information included was based on the following hierarchy: DHHS is offered if available, then IARC, then EPA. Failure of these organizations to evaluate a chemical does not necessarily mean that the chemical does not cause cancer.

Reproductive and Developmental Effects

Information about reproductive and developmental effects was obtained primarily from three data files that are included in TOMES Plus, a proprietary database of Micromedex, Inc., Denver, CO. These data files are Reprotext, edited by Betty J. Dabney, PhD; the Teratogen Information System (TERIS), developed by the University of Washington; and *Shepard's Catalog of Teratogenic Agents*, written by Thomas H. Shepard, MD. An additional source of information was *Reproductive and Developmental Toxicants*, a 1991 report published by the U.S. General Accounting Office (GAO Report no. GAO/PEMD-92-3) that lists 30 chemicals of concern because of widely acknowledged reproductive and developmental consequences. The 30 chemicals are alcohol, arsenic, cadmium, carbon disulfide, carbon monoxide, chlordecone, chloroprene, DDT, DBCP, DES, ethylene dibromide, EGEE, EGME, ethylene oxide, gossypol, hexachlorobenzene, lead, lithium, mercury, nicotine, PBBs, PCBs, 2,4,5-T, TCDD, tobacco smoke, toluene, vinyl chloride, vitamin A, and warfarin.

The topic of reproductive hazards is controversial and emotionally charged. Potentially high risk to the fetus may warrant considering termination of the pregnancy. Most clinicians are not adequately prepared to help the patient make this decision. Expert assistance may be available from regional poison control centers, regional reproductive risk/teratogen information centers, or the Motherisk Program. For more information, see *Appendices I* and *III*.

Case Study

On Tuesday afternoon, a 52-year-old man with previously diagnosed coronary artery disease controlled by nitroglycerin describes episodes of recurring headache for the past 3 weeks. Mild nausea often accompanies the headache; there is no vomiting. He describes a dull frontal ache that is not relieved by aspirin. The patient states that the headaches are sometimes severe; at other times they are a nagging annoyance. The durations range from half an hour to a full day. His visit was prompted also by a mild angina attack that he suffered this past weekend, shortly after awakening on Sunday morning. He has experienced no further cardiac symptoms since that episode.

History of previous illness indicates that the patient was diagnosed with angina pectoris 3 years ago and has been taking nitroglycerin 0.4 mg sublingually prophylactically before vigorous exercise. He also takes one aspirin every other day. He has been symptom-free for the past 2 ½ years. Sublingual nitroglycerin relieved the pain of the Sunday morning angina attack within several minutes. The patient does not smoke and rarely drinks alcohol.

He is a trim man with a slightly ruddy complexion. At present, he is afebrile, and his vital signs are blood pressure 120/85, pulse 80, respirations 20. Physical examination including HEENT, heart, lungs, and neurologic exam is normal. The results of an ECG with a rhythm strip performed in your office are unremarkable. Subsequent laboratory testing reveals normal blood lipids, cardiac enzymes, CBC, sedimentation rate, glucose, creatinine, and thyroid function.

(a) What would you include in the patient's problem list?

(b) What would you include in the differential diagnosis?

(c) What additional information would you seek to assist in the diagnosis?

Introduction

The preceding case study describes a patient with angina. He has new, nonspecific symptoms of headache and nausea. Suppose this patient lived near a hazardous waste site. Would your differential diagnosis change? If the patient refinished furniture as a hobby, would you consider this important? Is there a connection between his headaches and cardiac symptoms? How would you investigate the possible correlation? Could he be exposed to chemicals in his workplace? Each of these factors could play a role in the etiology of this patient's illness; each exposure could cause disease.

The patient described in the case study–a 52-year-old male with angina–is portrayed in three different scenarios throughout this document. An exposure history form, completed by the patient in each scenario, provides clues that prompt the clinician to investigate the possibility of toxic exposure.

- ***Scenario 1: This patient is an accountant who has had the same job and residence for many years.***
- ***Scenario 2: This patient owns a commercial cleaning service and uses cleaning products at various industrial and commercial sites.***
- ***Scenario 3: This patient is a retired advertising copywriter who lives in the vicinity of an abandoned industrial complex.***

Most environmental and occupational diseases either manifest as common medical problems or have nonspecific symptoms. It is the etiology that distinguishes a disorder as an environmental illness. Unless an exposure history is pursued by the clinician, the etiologic diagnosis may be missed, treatment may be inappropriate, and exposure can continue.

Most people with illness caused or exacerbated by exposure to hazardous substances obtain their medical care from clinicians who are not specialists in either environmental or occupational medicine. Few clinicians, however, routinely elicit information about the home, workplace, or community environment as part of the demographic and social history. In a study of a primary care practice in an academic setting, only 24% of 625 charts had any mention of the patient's occupation. Only 2% of the charts had information on exposures, duration of present employment, and past occupations. In addition, clinicians caring for adolescents seldom ask about their work exposure and history during routine health care visits or when evaluating symptoms.

Although many clinicians do recognize the importance of taking a work and exposure history for evaluating certain problems, most have had little training or practice in doing so. Extensive knowledge of toxicology is not needed to diagnose environmental and occupational disease. The same criteria are employed as those used in diagnosing other medical problems–history including onset and temporal pattern of symptoms, palliative and provocative factors, physical examination, and laboratory results. If necessary, consultation with industrial hygienists or environmental testing can be used. In addition to current exposures, the clinician must consider the long-term or latent effects of past exposures to agents such as asbestos, radiation, and chemical carcinogens.

Investigating environmental and occupational illness is illustrated in this monograph. The aim is not to demonstrate all exposure possibilities but rather to illustrate the principles and the *process* of investigating this etiology. The exposure history form (pages 23-26), which can be completed by the clinician or by the patient (to save staff time), will guide the clinician through various aspects of this process. The form elicits many important points of an exposure history including job descriptions and categories associated with hazardous substances, physical and biologic agents, and temporal and activity patterns related to environmental and occupational disease. The form explores past and current exposures.

Taking an exposure history requires only a few minutes of the clinician's time and can be abbreviated, expanded, or focused according to the patient's signs and symptoms. The exposure history form is designed for quick scanning of important details and can be copied and used for a permanent database as well as for the investigation of current problems.

The diagnosis of environmental or occupational disease cannot always be made with certainty. Sound clinical judgment must be used, and common etiologies should be considered. The multifactorial nature of many conditions, particularly chronic diseases, must not be overlooked.

An exposure history should be taken on every patient. It is of particular importance if the patient's illness occurs at an atypical age or is unresponsive to treatment.The clinician must also keep in mind that many organ systems are affected by toxic exposure (Table 1). The latency period from exposure to manifestation of disease can vary–ranging from immediate to delayed (hours or days) to prolonged (decades).

With practice using the exposure history form and a network of referrals, the primary care clinician can play an important role in detecting, treating, and preventing disease resulting from toxic exposures.

Organ Systems Affected by Toxic Exposure

The respiratory system is both a target organ and a portal of entry for toxicants. Adult-onset asthma and death from asthma are increasing. More than 100 toxicants are known to cause asthma, and many more can exacerbate it.

Irritant and allergic contact dermatitis account for 90% of occupational skin disorders. Other skin disorders with exposure etiologies include pigment alterations, chloracne, urticaria, and malignant neoplasms.

Alcohol abuse is a potential confounding factor in the evaluation of patients with suspected toxic exposure. However, a history of alcohol use does not necessarily exclude an environmental or occupational etiology. Symptoms of liver disease due to toxic exposure can mimic viral hepatitis.

About 4000 new cases of renal disease of unknown etiology are diagnosed annually. Organic solvents and heavy metals are two classes of toxicants known to adversely affect renal function.

Neurotoxins can cause peripheral neuropathy, ataxia, parkinsonism, seizures, coma, and death. Many chemicals cause mild central nervous system depression that may be misdiagnosed as personality disorders or that can progress to psychoses or dementia. Sensory impairment can also be caused by exposure to toxicants (e.g., visual disturbances caused by methanol) and physical agents (e.g., hearing impairment caused by loud noise).

About 200,000 infants are born annually with some form of birth defect. The causes of most of these defects are unknown.

Table 1. Organ systems often affected by toxic exposure

Organ/System	Exposure Risks
Respiratory	asbestos,* radon,* cigarette smoke, glues
Dermatologic	dioxin,* nickel, arsenic,* mercury,* cement (chromium*), PCBs,* glues, rubber cement
Liver	carbon tetrachloride,* methylene chloride,* vinyl chloride*
Kidney	cadmium,* lead,* mercury,* chlorinated hydrocarbon solvents*
Cardiovascular	carbon monoxide, noise, tobacco smoke, physical stress, carbon disulfide, nitrates,* methylene chloride*
Reproductive	methylmercury,* carbon monoxide, lead,* ethylene oxide
Hematologic	arsenic,* benzene,* nitrates,* radiation *
Neuropsychologic	tetrachloroethylene,* mercury,* arsenic,* toluene,* lead,* methanol,* noise, vinyl chloride*

*This substance is covered in *Case Studies in Environmental Medicine*, which is a series of self-instructional booklets on specific chemical hazards developed by the Agency for Toxic Substances and Disease Registry (ATSDR), Division of Health Education. A complete list of titles and information on how to obtain them is on page 38.

The cardiovascular and hematologic systems are frequent targets of toxicants. Cardiovascular changes, as well as exacerbation of preexisting cardiovascular conditions, can result from exposure to noise and to chemicals such as carbon monoxide and tobacco smoke. Benzene can cause bone marrow changes leading to aplastic anemia, acute leukemia, and chronic myelogenous leukemia.

Toxicants in the Home/Environment

The clinician should consider the following sources, which are discussed below, when eliciting information on exposures in the home and environment:

- Indoor air pollution
- Common household products
- Pesticides and lawn care products
- Lead products and waste
- Recreational hazards
- Water supply
- Soil contamination

Indoor Air Pollution

Tobacco Smoke

Environmental tobacco smoke is a mixture of more than 4700 compounds. Mainstream smoke is exhaled by the smoker, and sidestream smoke comes off the smoldering end of the cigarette and is inhaled by adjacent persons (passive smokers). Sidestream smoke contains more carcinogenic hydrocarbons and respirable particles than mainstream smoke. All smokers should be encouraged to stop smoking; if household members will not refrain from smoking, they should smoke only in well-ventilated or isolated areas.

Does anyone in the household smoke? How many packs per day?

Wood Stoves/Gas Ranges

Thirteen million wood stoves are in use in the United States, and 800,000 are sold annually. When not properly maintained and vented, wood stoves emit noxious gases including carbon monoxide, oxides of nitrogen, particulates, and hydrocarbons. Studies have shown that children living in homes heated with wood stoves have a significant increase in respiratory symptoms compared with children living in homes without wood stoves.

Does the patient have a wood stove?

Is there a smoke smell indoors?

When was the last time the chimney and stove were cleaned?

Gas ranges, which may produce nitrogen oxide, a respiratory irritant, are used for cooking in more than half of U.S. homes. In low-income areas, gas stoves may be used not only for cooking but as a supplemental source of heat. Proper ventilation and routine inspection and maintenance are necessary in residences where wood or gas stoves are used.

If the patient uses a gas range, is it in proper working order?

Does the patient use the gas range for heat?

Building Materials

Building materials, home improvement products, and textiles used in the home can pose health risks. For example, formaldehyde volatilizes

Does the patient live in a mobile home?

Was urea formaldehyde foam used for insulation?

Is cabinetry or furniture made of pressed wood?

from particle board, insulation materials, carpet adhesives, and other household products. This is a particular problem in the confined spaces of mobile homes. Formaldehyde exposure can cause rhinitis, nausea, dry skin or dermatitis, and upper respiratory and eye irritation. It has also been reported to precipitate bronchospasm in persons who have asthma.

Asbestos

Was asbestos insulation used on pipes or hot water tank?

Do walls and ceilings have sprayed-on or troweled-on material?

Is renovation work planned in any of the areas containing asbestos?

Are adults in the household exposed to asbestos on the job?

Asbestos was widely used from 1950 to the early 1970s in areas requiring sound proofing, thermal proofing, or durability (e.g., floor and ceiling coverings, heating and water pipe insulation). It was often applied as a spray-on material. Asbestos that is in good condition and not respirable is generally not a risk. However, when it becomes frayed or friable (i.e., easily crumbled), asbestos fibers can be released into the air. Exposure to these fibers has been associated with lung cancer, asbestosis, and mesothelioma. The occurrence of disease is influenced by type of asbestos mineral inhaled, concentration and dimension of the fibers, and exposure duration. In 1986, the Environmental Protection Agency (EPA) estimated that friable asbestos may be present in as many as 35,000 schools in the United States, potentially exposing 15 million schoolchildren and 1.4 million adults. Smoking cigarettes, in addition to asbestos exposure, increases the risk of cancer by an order of magnitude above smoking alone or asbestos exposure alone. Children may be at greater risk than adults because of their long life expectancy, high activity rates, high breathing rates, more time spent near the floor where fibers accumulate, and greater likelihood of contact (through curiosity or mischief). (For further information on the health hazards of asbestos exposure, consult *Case Studies in Environmental Medicine: Asbestos Toxicity*, ATSDR, June 1990.)

Radon

Has the patient's home been tested for radon?

If yes, what were the results?

Are there high levels in homes in the area?

Do children spend a significant amount of time in the basement or on the first floor of the home, where radon might tend to be in higher concentrations?

Radon, a colorless, odorless gas, is a decay product of uranium found in significant concentrations in some areas. Radon itself does no harm, but its progeny attach to airborne particulates such as cigarette smoke and can be inhaled. During subsequent decay, the progeny emit high-energy alpha particles that may injure adjacent bronchial cells, thereby causing lung cancer. Five to ten percent of single-family homes in the United States have been estimated to exceed the EPA radon recommended guideline of 4 picocuries per liter of air. EPA estimates that approximately 14,000 lung cancer deaths per year are attributable to radon. (For further information about radon exposure and its health effects, see *Case Studies in Environmental Medicine: Radon Toxicity*, ATSDR, September 1992.)

Common Household Products

Does the patient use any of the following on a regular basis: cleaners for glass, oven, floors, drains, toilets, polishes, air fresheners and disinfectants, glues, solvents, paint strippers, sealants?

A 1987 EPA study found approximately 12 common organic pollutants in concentrations 2 to 5 times higher in air inside homes than in outdoor air from use of household products. Product warning labels are often inadequate and pertain to acute exposures only. Long-term or repeated use of some household chemicals, such as chlorinated hydrocarbons, can result in cancer. Commonly used compounds that

can have serious adverse effects are methylene chloride (found in paint strippers and thinners, and adhesive removers), tetrachloroethylene (used in dry cleaning of clothes), and paradichlorobenzene (found in room air fresheners, toilet bowl deodorizers, and moth crystals). (See *Case Studies in Environmental Medicine: Methylene Chloride Toxicity*, ATSDR, June 1990, and *Tetrachloroethylene Toxicity*, ATSDR, June 1990.)

Where are these chemicals stored and disposed of?

Pesticides and Lawn Care Products

Pesticides and lawn care products are potentially hazardous, especially to children. Pesticide exposure can occur through dermal contact, inhalation, or ingestion. At least 1400 active ingredients can be found in more than 34,000 available preparations of insecticides, herbicides, fungicides, and other antibiologic preparations. These agents have different mechanisms of action and toxicity. Estimated annual use of these chemicals is 2.6 billion pounds.

Despite the ban on certain pesticides in the United States, exposure can still occur through improper use, storage, and disposal. Some banned pesticides are used in foreign countries and may return to this country on imported foods. Proper use and storage of household pesticides and proper cleaning of food, especially raw fruits and vegetables, can help protect consumers.

Does the patient use pesticides on the garden and lawn?

Does the patient employ a professional lawn-care company?

Are children allowed to play in areas recently sprayed with pesticides or lawn-care products?

Does the patient use bug repellants?

Does the patient know what to do in case of accidental poisoning?

Lead Products and Waste

Lead poisoning continues to be a significant health problem in the United States. Although lead was banned from paint for home use in 1972, millions of homes, particularly those built before 1950, still contain high amounts of lead in paint that is peeling and accessible for ingestion by children. Lead exposure also occurs through drinking water, especially in homes that have lead plumbing or lead-soldered pipes. Significant exposures have occurred in children who played in lead-contaminated soil. Acidic foods, such as juices, stored in imported pottery may leach lead from ceramic glazes. Some ceramic glazes used by hobbyists also may contain lead. Air can be contaminated with this metal through use of leaded gasoline. Parents can inadvertantly bring it home on their clothing and shoes, or in their cars if they work in jobs where they are exposed to lead dusts or lead-containing compounds.

More than a million U.S. workers are potentially exposed to lead daily in hundreds of occupations such as construction work, radiator repair, metals recycling, battery manufacturing, smelting, and pigments formulating. Good workplace and personal hygiene practices can prevent the majority of these "take-home" exposures.

The 1985 intervention level of 25 µg/dL has been revised downward to 10 µg/dL. Childhood lead exposure has been associated with lower class ranking and higher absenteeism in school, poor eye-hand coordination, slow reaction time, and lower vocabulary test scores. Consequences of childhood lead exposure have been shown to endure into adulthood. (See *Case Studies in Environmental Medicine: Lead Toxicity*, ATSDR, Revised September 1992.)

What year was the patient's home built? Is indoor paint in poor repair?

Is the inside of the patient's home being renovated?

Has the patient's drinking water been tested for lead?

Does the patient use imported earthenware pottery?

Do any household members work with lead (e.g., in a lead refinery or smelter, battery factory, or power plant)? If *yes*, are work clothes brought home?

Do any household members work with arts and crafts products containing lead?

Does the patient live near a lead refinery or smelter, battery factory, or power plant?

Recreational Hazards

Do the patient's children play on wooden playground equipment that has been treated and sealed?

Do the children play in a sandbox that may contain tremolite (asbestos)?

Recreational areas and products can pose a hazard to health. Fishing and swimming in contaminated lakes and streams can expose participants to toxins contained in polluted waters. Wooden playground structures that have not been treated with protective sealants may allow children to have dermal contact with potentially hazardous wood preservatives; these include arsenic-containing compounds, pentachlorophenol, and creosote. Some play sands and clays have been reported to contain asbestos-like fibers. Other materials used in arts and crafts involve potentially hazardous silica, talc, solvents, and heavy metals such as lead and cadmium. Toxic materials may be encountered in making stained glass and jewelry, woodworking, model building, and oil and airbrush painting. One need not be directly involved in these activities to become exposed; merely being in the vicinity of a work area may cause exposure. Federal legislation (Labeling of Hazardous Materials Act) will require that all chronically hazardous materials be labeled as inappropriate for children's use. (See ATSDR series *Case Studies in Environmental Medicine: Arsenic Toxicity,* June 1990; *Pentachlorophenol Toxicity*, December 1992; *Cadmium Toxicity*, June 1990; and *Asbestos Toxicity*, June 1990.)

Water Supply

What is the source of the patient's water supply?

If the patient uses a private well, when was the last time the water was tested?

Both public water supplies and private wells can be a source of toxic exposure, especially for industrial solvents, heavy metals, pesticides, and fertilizers. For example, an EPA groundwater survey detected trichloroethylene in approximately 10% of the wells tested. It is estimated to be in 34% of the nation's drinking water supplies. Up to 25% of the water supplies have detectable levels of tetrachloroethylene. Methylene chloride may remain in groundwater for years. Some solvents can volatilize from showers and during laundering of clothes, thereby creating risk of toxicity via inhalation. Nitrates, a common contaminant of rural shallow wells, pose a risk of methemoglobinemia, especially to infants. *(See* ATSDR series *Case Studies in Environmental Medicine: Asbestos Toxicity*, June 1990; *Arsenic Toxicity,* June 1990; *Lead Toxicity,* Revised September 1992; *Nitrates/Nitrites Toxicity*, October 1991; *Trichloroethylene Toxicity*, January 1992; *Methylene Chloride Toxicity,* June 1990; *Tetrachloroethylene Toxicity,* June 1990.)

Soil Contamination

Did the patient or previous owners use chlordane or other pesticides or termiticides in the home?

What is the history of the site on which the home was built?

Ingestion of contaminated soil poses a risk of toxicity, especially to children under the age of six because of natural mouthing behaviors. Lead is a common soil contaminant. Dioxin also adsorbs to soils. Certain pesticides such as chlordane can remain in the soil for years. (See ATSDR series *Case Studies in Environmental Medicine: Arsenic Toxicity*, June 1990; *Lead Toxicity*, Revised September 1992; *Dioxin Toxicity*, June 1990; *Chlordane Toxicity*, December 1992; *Cadmium Toxicity*, June 1990; *Chromium Toxicity*, June 1990.)

Using the Exposure History Form

A work and exposure history has three components: *Exposure Survey, Work History*, and *Environmental History*. The main aspects of an exposure history (summarized in Table 2) will be elicited through the exposure history form (pages 23-26). Although a positive response to any question on the form indicates the need for further inquiry, a negative response to all questions does not necessarily rule out a toxic exposure etiology or significant previous exposure.

All patients should complete exposure history forms, although the form need not be evaluated extensively in every clinical situation. As in all data-gathering activities, sound clinical judgment must be exercised.

Table 2. Components of an exposure history

Part 1. Exposure Survey

A. Exposures

- Current and past exposure to metals, dust, fibers, fumes, chemicals, biologic hazards, radiation, noise, vibration
- Typical work day (job tasks, location, materials, agents used)
- Changes in routines or processes
- Other employees or household members similarly affected

B. Health and Safety Practices at Worksite

- Ventilation
- Medical and industrial hygiene surveillance
- Employment exams
- Personal protective equipment (e.g., respirators, gloves, coveralls)
- Lockout devices, alarms, training, drills
- Personal habits (Smoke, eat in work area? Wash hands with solvents?)

Part 2. Work History

- Description of all prior jobs including short-term, seasonal, part-time employment and military service
- Description of present job(s)

Part 3. Environmental History

- Present and prior home locations
- Jobs of household members
- Home insulating, heating and cooling system
- Home cleaning agents
- Pesticide exposure
- Water supply
- Recent renovation/remodelling
- Air pollution, indoor and outdoor
- Hobbies: painting, sculpting, welding, woodworking, piloting, autos, firearms, stained glass, ceramics, gardening
- Hazardous wastes/spills exposure

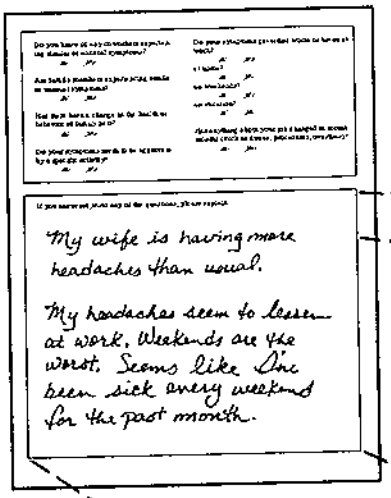
Part 1. Exposure Survey

Past and current exposures are recorded on pages 1 and 2 of the exposure history form, which is designed for easy completion by the patient and quick scanning for pertinent details by the clinician. The questions investigate the following: known exposure to metals, dust, fibers, fumes, chemicals, physical agents, and biologic hazards; details about known toxicant exposure; other persons affected; temporal patterns and activities, changes in routines and worksite characteristics, and protective equipment use.

If the patient answers *yes* to one or more questions on Part 1, the clinician must follow up by asking the patient progressively more detailed questions about the possible exposure. Special attention should be directed to the route, dose, duration, and frequency of any identified exposure.

Scenario 1 below illustrates the use of part 1 of the form with the patient described in the case study (page 1). The patient's chart reveals that he has worked as an accountant in the same office for the past 12 years. On the completed form, he indicates that no other workers are experiencing similar or unusual symptoms, and he denies recent changes in his job routine. The patient answered *yes* to three questions: "Are family members experiencing the same or unusual symptoms?"; and "Do your symptoms get either worse or better at work? on weekends?" His explanations of these answers reveal a possible temporal relationship between his symptoms and home. The clue and the clinician/patient dialogue follow.

Scenario 1:
52-year-old male accountant with angina
Chief complaint: headache and nausea



If you answered *yes* to any of the questions, please explain.

My wife is having more headaches than usual.

My headaches seem to lessen at work. Weekends are the worst. Seems like I've been sick every weekend for the past month.

Clinician: I see that you noted that your wife is having headaches.
Patient: Yes. She has frequent headaches. In the last 3 or 4 weeks she has had more than usual. She usually has one every month or so; this past month she had three.

Clinician: You also state that your headaches are worse on weekends.
Patient: Yes, they seem to be. If I wake up on a Saturday or Sunday with a headache, it usually gets worse as the day progresses. In fact, that's usually when I feel nauseated too.

Clinician: Do your symptoms seem to be aggravated by certain activities around the home? A hobby or task?
Patient: No, I usually wake up with the headache. I don't think there's a connection with anything I do.

Clinician: Do your symptoms change at all at work?
Patient: Now that you mention it, if I wake up with a headache, by the time I get to work–it takes about 25 minutes–the headache is usually gone.

Clinician: Your angina attack occurred on a Sunday morning. Describe your weekend leading up to the attack.

Patient: It was a fairly quiet weekend. We had dinner at home Friday evening and just relaxed. On Saturday I spent the day packing old books and storing them in the attic and chopping and stacking firewood. I took one nitroglycerin tablet before doing the heavy work, at about 2 PM. Saturday night we had friends over for dinner. We had a fire in the fireplace and visited until about 11 PM. I had one glass of wine with dinner. I was beginning to feel a little stiff and sore from the work I did that afternoon. Sunday morning I woke up with a headache again. A few minutes after awakening, while I was still in bed, I had the attack. It was mild, not the crushing pain I've had in the past. I had the headache all day.

The preceding dialogue reveals that the patient's symptoms may be associated with the home environment and his cardiac symptoms, headache, and nausea may be related. His symptoms seem to be exacerbated at home and lessen at work. Further questioning is needed to pursue this lead.

Clinician: What does your wife do for a living?
Patient: She's an attorney.

Clinician: Do either one of you have a hobby?
Patient: My hobby is photography. My wife is an avid gardener.

Clinician: Do you have your own darkroom?
Patient: No, I occasionally use a friend's. For the past year I've had my film and prints processed commercially.

Clinician: Does your wife use any pesticides or chemicals in the garden?
Patient: No, she does strictly organic gardening and uses only natural means of pest control.

Clinician: Do you work on your car?
Patient: No.

Clinician: Have you gotten any new furniture or remodeled your home in the past few years?
Patient: No.

Clinician: What is your source of heating and cooking in the home?
Patient: We have a natural gas, forced-air heating system. We cook with gas and use the fireplace a lot in winter.

Clinician: How long have you lived in this home and how old is your furnace?
Patient: We've lived there for 23 years. The furnace was replaced about 12 years ago.

Clinician: I see that you recently insulated your home. What exactly did you do?
Patient: Yes. Last month I added extra insulation to the attic, insulated the crawl space, replaced all the windows with double-paned windows, and weatherized all doorways.

Clinician: Have you noticed that the headaches coincide with days you have used the fireplace?
Patient: There could be a connection. I definitely use the fireplace more on weekends. This past Saturday I had a fire blazing all day.

A temporal relationship between the headaches and being in the home has been revealed. Some sources of toxicants have been eliminated (formaldehyde and other volatile organic chemicals from new furniture and rugs; toxic chemicals used in hobbies or gardening). A correlation may exist between symptoms and use of the fireplace. The fireplace could increase negative pressure in the house, causing backdrafting of furnace gases. The furnace is old; it may be malfunctioning or producing excessive carbon monoxide. The patient's symptoms, including his angina attack, would be consistent with carbon monoxide poisoning.

Although the patient's symptoms could be associated with his preexisting disease, evidence is strong enough at this point to investigate the possibility of environmental exposure. Contacting the local gas company to request that they check the furnace and stove for malfunctions and leaks would be appropriate. The fireplace should be checked for proper drafting and for deposits of creosote in the chimney.

A carboxyhemoglobin (COHb) level on the patient may confirm carbon monoxide poisoning. The patient should be advised to ventilate the

house until the furnace is checked or to stay out of the house until the gas company deems it safe. Symptoms of headaches usually do not occur below 15% COHb, but the half-life of COHb is only several hours.

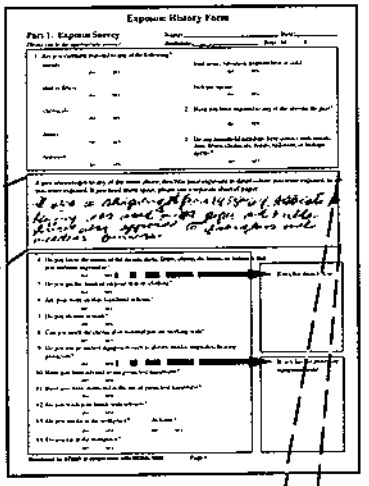
A COHb level performed on this patient is reported to be 6%, which is high for a nonsmoker. The gas company discovers a cracked heating element in the 12-year-old furnace, which resulted in carbon monoxide fumes circulating throughout the house. The use of the fireplace most likely increased the backdrafting of fumes. The furnace is replaced, the exposure ceases, and the patient's symptoms abate. He experiences no further cardiac symptoms.

It is not necessary to understand the jargon of a particular trade; persistent questioning by the clinician can clarify the tasks involved and reveal possible exposures.

The exposure history form may also alert the clinician to past exposures. Most often, neither the job title nor the patient's initial description of job duties reveals clues of exposure. It is usually helpful to have a patient describe a routine work day, as well as unusual or overtime tasks. Patients tend to use jargon when describing their jobs. It is the clinician's challenge to persistently question the patient to elucidate possible exposures; it is not necessary to have foreknowledge of a particular trade. Start with general questions and work toward the more specific.

Page 1 of the form reveals another clue–this patient was exposed to asbestos about 30 years ago. The questioning that the clinician conducts, despite having neither knowledge of the patient's trade nor understanding of the jargon, follows.

Scenario 1:
52-year-old male accountant with angina
Chief complaint: headache and nausea


Exposure History Form

If you answered *yes* to any of the items above, describe your exposure in detail—how you were exposed; to what you were exposed.

I was a shipwright from 1958-64. Asbestos lagging was used on the pipes and hulls. I was also exposed to fiberglass and welders fumes.

Clinician: You state here that you were exposed to asbestos, fiberglass, and welders' fumes way back in '58.
Patient: Yes, during my days as a shipwright.

Clinician: Did you actually handle the asbestos?
Patient: No, the pipe laggers were the tradesmen that handled the asbestos. Oh, you might be setting a bracket or plate next to a pipe and accidentally hit the pipe and dislodge some asbestos, but otherwise, shipwrights didn't handle it. You only had asbestos where there were steamlines from the boiler carrying high-pressure steam to other units like a winch or an auxiliary motor.

Clinician: What does a shipwright do? What was a routine day for you?
Patient: There was no routine day. The shipwrights were the cream of the journeymen crop; we did everything from outfitting, to establishing the cribbing on the launching gang, to shoring. I worked on the outfitting docks. We did ship reconversions. I did a lot of work on the forepeak and hawse pipes when I wasn't working below decks.

Clinician: What exactly were your tasks below decks?
Patient: Most transporters were converted to passenger ships after the war; there was a lot of shifting of equipment and pipes. Basically, the ships were gutted. They would be completely revamped. The shipwrights would do all the woodworking, finish work, plates, and so on. Then, when everything was in place, it would be insulated and the pipes would be lagged.

Clinician: So you worked throughout the ship? And when you finished your tasks the laggers would come in?
Patient: No, no. There might be ten different tradesmen working in an afterpeak at one time. You'd be working next to welders, flangers, pipefitters, riveters, laggers; you name it. These conversions were done round-the-clock, 7 days a week; it could take a year and a half to complete a conversion. All the tasks were being done simultaneously.

Clinician: How long would the lagging take?
Patient: The lagging could take 6 to 10 months; sometimes longer. They were constantly cutting these sections of asbestos to fit the pipes. Then they would attach the sections with a paste and wrap it with asbestos wrapping.

Clinician: Could you see the asbestos in the air?
Patient: Oh yes. Sometimes it was so thick you couldn't see 5 feet in front of you. It was white and hung in the welders' fumes like smog.

Clinician: Did you use any protective equipment? Masks, respirators?
Patient: No. Nobody ever said it was dangerous. We were bothered more by the fiberglass and welders' fumes than anything. We thought

fiberglass was more dangerous because it was itchy and caused a rash. The air was blue from the welding fumes; if you worked in that for a year, you knew it was affecting you. It inspired me to go back to school and get my accounting degree. But we were blue-collar workers; we were more concerned with welders' flash, a boom breaking, or someone getting crushed between plates than we were with asbestos.

Clinician: You worked as a shipwright for 6 years?

Patient: Yes, about that. Five of those years as an outfitter on conversions.

The dialogue in which the clinician engaged the patient neither determines whether the patient's asbestos exposure was significant, nor does it confirm that he suffered adverse effects from the exposure. It is merely a starting point for investigation. The questioning establishes that approximately 30 years ago this patient received a possibly severe exposure to asbestos fibers for a duration of 5 or 6 years. Because quantitative data on this patient's exposure is impossible to obtain, a qualitative description ("Sometimes it was so thick you couldn't see 5 feet in front of you") can facilitate assessment of the exposure when consulting with an occupational medical specialist (see Appendix). In this scenario, the disclosure should prompt the clinician to monitor the patient closely for early detection of treatable health effects from asbestos exposure. A chest X ray would be advised and pulmonary function tests should be considered. Vaccination for influenza may be warranted, depending on the results of the chest X rays. Consulting an occupational medical specialist could help determine the best way to evaluate and treat this patient.

An exposure history may suggest the need for periodic monitoring by alerting the clinician to a past exposure.

In this scenario, the clinician has successfully diagnosed an illness due to an environmental toxic exposure (carbon monoxide) and has noted a significant past exposure (asbestos), which needs follow-up. Had the clinician failed to pursue an exposure history, the patient's current illness might have been misdiagnosed, treatment might have been inappropriate, or measures might not have been implemented to prevent further carbon monoxide exposure leading to a risk of continued progression of the angina, as well as coma and death involving other household occupants.

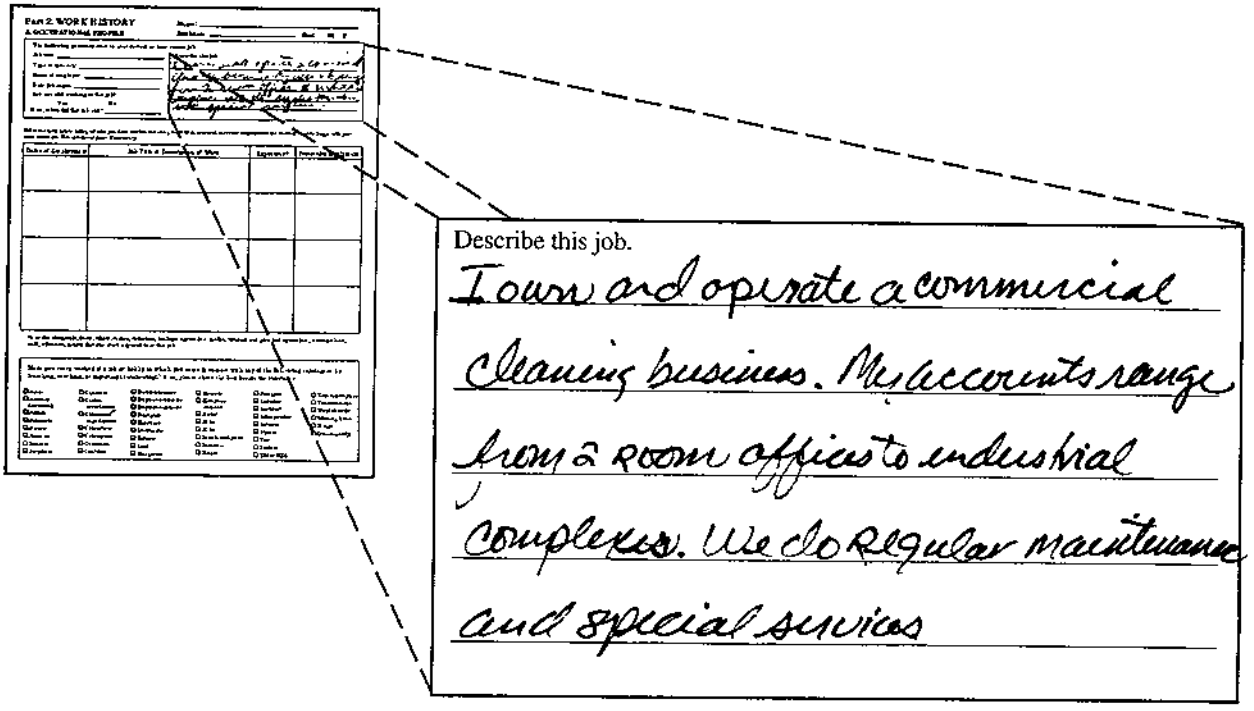
Part 2. Work History

Part 2 of the exposure history is a comprehensive inventory of the patient's occupations, employers, and current and potential exposures in the workplace. No questions on allergies and principal symptoms have been included on the presumption that the clinician will provide more detail elsewhere in the medical record.

In evaluating Part 2 of the form, the clinician should note every job the patient had, regardless of duration. Information on part-time and temporary jobs could provide clues to toxic exposure. Details of jobs may reveal exposures unexpected from the job titles. Asking if any processes or routines have been changed recently can be helpful. Military service may have involved toxic exposure.

Scenario 2 below involves another instance of a 52-year-old male with angina as described in the case study (page 1); he suffered an angina attack and complains of recurring headaches and nausea. This patient is the owner of a commercial cleaning service. He performs some of the cleaning himself. Scanning pages 1 and 2 of the form, the clinician notes that, in his work, the patient is exposed to cleaning chemicals including detergents, ammonia, and cleansers. The patient does not notice any temporal relationship of symptoms to activity. Questioning the patient extensively about the cleaning products fails to yield any suspicious exposure possibilities. Perusal of *Part 2. Work History*, however, reveals another clue. The clinician's investigation follows.

Scenario 2:
52-year-old male owner of a commercial cleaning service
Chief complaint: headache and nausea



Clinician: You own a commercial cleaning service?
Patient: Yes, I've been in business for 10 years.

Clinician: Do you do the cleaning yourself?
Patient: I don't do as much as I used to. I have a crew of about six full-time employees. I do more managing than cleaning but I have been known to roll up my sleeves and pitch in when need be.

Clinician: You clean residences and commercial businesses?
Patient: Yes, I have 20 residential accounts and 15 commercial accounts.

Clinician: What are the commercial accounts?
Patient: The downtown administrative offices of the school district, several realty offices downtown, and the business offices of the viscose rayon mill. I have six accounts in the Shaw Building downtown–small medical offices–and five retail stores in the Hilltop Mall.

Clinician: So your headaches have been occurring for about 3 weeks now? Have there been any changes in your routine–work or otherwise–in the last 3 weeks?
Patient: I've worked more hours than usual. I've been doing a special project for the rayon mill. They built new offices. We moved all the old offices into the new building. That has entailed cleaning and moving furniture, files, books, and exhibits. It's been tedious. Fortunately, most of the staff has been either out on vacation or at an international conference in Europe; so the building has been empty. We've been able to set our own pace and come and go any day or time that suits us, so long as we clear it with security.

Clinician: Are any other workers having similar symptoms?
Patient: No, nobody else has complained about feeling sick.

Clinician: What exactly do they produce at that plant?
Patient: They make viscose–transparent paper. I used to work there during summers when I was in college. It was hot, hard work. And the whole place smelled like sulfur–rotten eggs. We used wood pulp cellulose, treated it with acids and other chemicals, and made cellulose filaments. I worked on the blending, ripening, and deaeration process.

Clinician: Can you smell the chemicals in the office building you're working in?
Patient: Some days there's a faint odor. Nothing like when I worked on the xanthating process. The business office building is on the northeast end of the complex. It's pretty remote from the processing plant.

Clinician: So how many extra hours have you worked the past 3 weeks?
Patient: Only about 10 hours each week. This past weekend I put in an extra 7 hours. I had to finish setting up the exhibits. I didn't trust the crew to handle the fragile exhibits, so I did the job myself.

Clinician: And on Sunday morning you had the angina attack. Tell me about your weekend leading up to the attack.
Patient: On Friday, I worked late setting up a huge model of the xanthating process. It was tedious work and I was sort of stressed by the time constraints to get the job done. I had broken a bottle from the exhibit when I disassembled the thing weeks ago. I was working especially carefully this time. On Saturday morning, I ran back to the plant to tie up all the loose ends and finish. In the afternoon, my wife and I spent several hours walking on the beach, despite an awful headache I had. We went to bed fairly early, about 10 PM. On Sunday morning, I had the attack. But the nitro helped almost immediately, and I had no other problems. It was pretty mild.

Clinician: What was in this bottle you broke?
Patient: I'm not sure, really. The bottle said carbon disulfide but the chemical did not smell like the carbon disulfide we used in the mill when I worked there. This stuff had a sweet odor. It was quite strong but it didn't have the nauseating rotten-egg smell of the plant.

Clinician: How did you clean it up?
Patient: I just soaked it up with rags and threw them out. The carpet dried fairly quickly.

Clinician: Did you get any of the chemical on you?
Patient: When the bottle fell and shattered, it soaked my pant leg and the toes of my shoes. I probably got some on my hands, too, when I cleaned it up.

Clinician: How much of the chemical was in the bottle? Did you report the accident to anyone at the plant?
Patient: The bottle was about a liter in size. It was full. No, I didn't report the accident. Frankly, I'm embarrassed about it. I thought I would just talk with the manager when he returns from Europe later this week.

Clinician: What did you do with the bottle?
Patient: I put the broken pieces in a paper bag and tossed it into my truck.

Clinician: Can you get it so we can read the label?
Patient: Sure. I'll call you as soon as possible.

The preceding conversation reveals a possible connection with the spill and this patient's symptoms. It warrants further investigation. The results of the patient's physical examination are normal.

The patient retrieves the broken bottle. The label on the bottle identifies the chemical–carbon disulfide–and the manufacturer. After obtaining permission from the patient, the clinician calls the manufacturer for information on carbon disulfide.

Clinician: My patient is a contract employee at a local textile company. In the process of his work he broke a bottle that was labeled carbon disulfide. He didn't report the accident and just cleaned it up himself. I am concerned that he may be experiencing health effects from the exposure.

Manufacturer: It would not surprise me. Carbon disulfide is dangerous stuff. Strict industrial controls are in effect to prevent exposure.

Clinician: He says the chemical did not smell like the carbon disulfide he remembered working with in the plant years ago. He says it had a sweet odor.

Manufacturer: The odor of the commercial grade used in the plant is altogether different from pure carbon disulfide, which I suspect was what was in the bottle he broke. Pure-grade carbon disulfide has a sweet odor.

Clinician: Can you send me information on carbon disulfide?

Manufacturer: Certainly. I'll send you a Material Safety Data Sheet on carbon disulfide today. I suggest that you report the accident to the safety manager at the textile plant.

The clinician receives a Material Safety Data Sheet on carbon disulfide (pages 29-30), reads the Health Hazard Data section, and discovers that this chemical can exacerbate cardiovascular disorders in persons receiving long-term exposure. Nausea and headache are among the acute effects of exposure, and primary routes of entry are inhalation and skin contact/absorption. Consultation with a toxicologist confirms that this patient's symptoms could indeed be caused by exposure to carbon disulfide. The clinician orders a CBC, ECG, urinalysis, tests of liver and kidney function, and determinations of COHb and electrolyte levels on this patient.

Air sampling in the office in which the incident occurred reveals airborne concentrations of 0.8 parts of carbon disulfide per million parts of air (0.8 ppm). The permissible exposure limit for an 8-hour time-weighted average is 4 ppm. The concentrations were most likely higher at the time of the incident 3 weeks ago. This indicates that besides the acute exposure the patient incurred at the time of the accident, he has been chronically exposed to carbon disulfide for the previous 3 weeks, although for a limited number of hours each week while driving with the contaminated rags and bottle in his truck.

Results of the laboratory tests on this patient, including the COHb level, all are within normal limits. The patient's exposure ceases, and he experiences no further symptoms. The clinician continues to monitor the patient's angina, which remains stable. Other employees at risk of exposure from this spill are also examined; none incurred acute exposure or suffered ill effects. At the suggestion of the clinician, the safety manager at the mill instructs the employees in proper safety practices and no further incidents occur.

Part 3. Environmental History

Part 3 of the exposure history form contains questions regarding the home and surrounding environment of the patient. Dialogue with the patient should include queries about the location of the house, water supply, and changes in air quality.

Proximity to industrial complexes and hazardous waste sites could cause residents' exposure to toxicants in the air, water, or soil. Community contamination is a growing public health concern; affected persons usually seek care first from their primary care providers. If a group of people with similar symptoms and exposures is identified, and an environmental exposure problem is suspected, the clinician should call the state health department or the federal Agency for Toxic Substances and Disease Registry at (404) 639-0615. (See Referral Resources, page 31, and the Appendix for more information.)

Are chemicals used in a well-ventilated place?

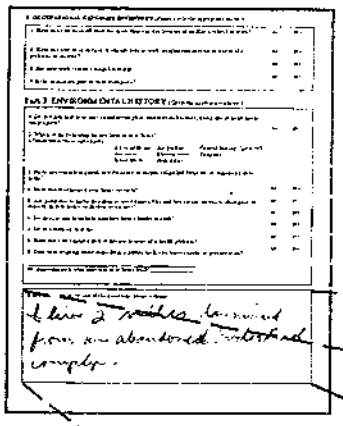
Is protective equipment used?

Hobbies are potential sources of toxicant exposure. For instance, model building, pottery-making, silk screening, gardening, stained-glass making, and woodworking all have been associated with hazardous exposure. Ask the patient what his or her hobbies are. All members in a household may be exposed to the hazardous substances from one person's hobby; small children may be especially susceptible.

Scenario 3:
52-year-old male, retired advertising copywriter with angina
Chief complaint: headache and nausea

Scenario 3 involves another patient described in the case study (page 1). In this scenario, the patient has been retired for 2 years; he took early retirement from a stressful job in advertising shortly after being diagnosed with angina. The patient's answers to the questions on the Exposure Survey (part 1 of the form) were *no*: he denies exposure to metals, chemicals, fibers, dust, radiation, and physical and biologic agents; he is not aware of a connection between his symptoms and activity or time; and to his knowledge other persons are not experiencing similar symptoms.

A clue appears on Part 3 of this patient's exposure history--the patient lives 2 miles from an abandoned industrial site and prevailing winds blow toward his house. In an effort to investigate this lead, the clinician initiates the dialogue that follows.



If you answered *yes* to any of the questions, please explain.

I live 2 miles downwind from an abandoned industrial complex.

Clinician: You state that you live several miles downwind from an abandoned industrial site. Do you know what chemicals might have been used at the site or what type of industry it was?
Patient: There was a fire at the site several weeks ago. The newspaper said that they used methylene chloride to make some kind of plastics. The firefighters found drums of methylene chloride buried on the property.

Clinician: Do you ever smell chemicals in the air?
Patient: Yes, in the mornings when the wind blows from that direction, I sometimes smell a sweet odor. My neighbors have mentioned it too. In fact, they told me that the smell is really strong when they do laundry or dishes, and when they shower.

Clinician: Have you smelled it in your water?
Patient: No.

Clinician: What is the source of your water?
Patient: I have city water, but my neighbors have a private well.

Clinician: Do you know if any agency is testing your neighborhood for contamination?
Patient: Not as far as I know.

The preceding dialogue has uncovered a possibility that the patient was exposed to a toxicant. Furthermore, this patient may represent an index case; others may also be exposed. To follow up this lead, the clinician contacts the state health department. The health department confirms that the site contains buried drums of methylene chloride and that it is under investigation.

An industrial hygienist employed by the health department informs the clinician that the methylene chloride can indeed exacerbate signs and symptoms of angina. The odor threshold for the chemical is 100 to 300 parts per million (ppm). An 8-hour exposure to 250 ppm methylene chloride can cause a COHb level above 8%.

The laboratory reports that the patient's COHb is 6%, indicating probable exposure to methylene chloride in this nonsmoker. The clinician calls the 24-hour consultation number ([404] 639-0615) of the Agency for Toxic Substances and Disease Registry (ATSDR), Emergency Response and Consultation Branch, for more information. The clinician is advised that COHb, which forms when methylene chloride metabolizes to carbon monoxide, can be detected in blood at levels of 4% to 9% when ambient air concentrations of methylene chloride are about 200 ppm. Many factors can influence body burden, including exposure level and duration, route of exposure, physical activity, and amount of body fat. A conference call with the emergency response coordinator, a toxicologist, an industrial hygienist, and a physician to

discuss the patient's signs and symptoms ensues. The clinician is given the local Association of Occupational and Environmental Clinics (AOEC) contact, who recommends a specialist who will provide follow-up care for this patient.

Results of the health department's tests of ambient air reveal no immediate crisis in the vicinity, although the levels are high; test results of water samples from private wells in the area are pending. ATSDR informs the regional office of the EPA of the situation. EPA provides immediate assistance to the affected area, clean-up is initiated, and threats to the surrounding population are mitigated.